What is claimed is:

1. A method of inhibiting hyperglycemia-induced or free fatty acid-induced reactive oxygen formation in a mammalian cell, the method comprising treating the cell with a pharmaceutically acceptable composition comprising GLP-1 (9-36) sufficient to inhibit the hyperglycemia-induced or free fatty acid-induced reactive oxygen formation in the cell.

2. The method of claim 1, wherein the reactive oxygen formation is hyperglycemia-induced.

3. The method of claim 1, wherein the cell is in a living mammal.

4. The method of claim 1, wherein the cell is selected from the group consisting of a nerve cell, a renal mesangial cell, a β cell, an adipocyte, an endothelial cell or a hepatocyte.

5. The method of claim 1, wherein the cell is an endothelial cell.

6. The method of claim 5, wherein endothelial cell is a vascular endothelial cell.

7. The method of claim 5, wherein the endothelial cell is in a mammal that has or is at risk for having diabetes, impaired glucose intolerance, stress hyperglycemia, metabolic syndrome, and/or insulin resistance.

8. The method of claim 5, wherein the mammal is critically ill.

9. The method of claim 5, wherein the mammal has chronic ischemia.

10. The method of claim 1, wherein the cell is a hepatocyte.

11. The method of claim 10, wherein the hepatocyte is in a living mammal that has or is at risk for ischemia/reperfusion injury, endotoxin injury, or alcoholic liver disease.

12. The method of claim 1, wherein the cell is a β cell.

13. The method of claim 12, wherein the β cell is in a living mammal that has or is at risk for impaired glucose-stimulated insulin secretion.

14. The method of claim 1, wherein the cell is a neuron.

15. The method of claim 14, wherein the neuron is a peripheral neuron.

16. The method of claim 1, wherein the cell is an adipocyte.

17. The method of claim 1, wherein the cell is a renal mesangial cell.

18. The method of claim 1, wherein the GLP-1 (9-36) has the sequence of SEQ ID NO:1.

19. The method of claim 1, wherein the GLP-1 (9-36) is an amide.

20. The method of claim 1, wherein the GLP-1 (9-36) further comprises an additional amino acid at the carboxy terminus.

21. The method of claim 20, wherein the additional amino acid is a Gly.

22. The method of claim 20, wherein the additional amino acid is an arginine.

23. The method of claim 1, wherein the GLP-1 (9-36) has the sequence of any one of SEQ ID NOs:2-16.

24. The method of claim 23, where the GLP-1 (9-36) further has an additional Arg at the carboxy terminus.

25. The method of claim 1, further comprising monitoring hyperglycemia-induced reactive oxygen formation after treatment with the GLP-1 (9-36) composition.

26. The method of claim 25, wherein the reactive oxygen formation is monitored by directly measuring reactive oxygen in the cell.

27. The method of claim 25, wherein the reactive oxygen formation is monitored by measuring prostacyclin synthase activity in the cell.

28. The method of claim 27, wherein the prostacyclin synthase activity is measured by measuring formation of 6-keto-$PGF_{1\alpha}$.

29. The method of claim 3, wherein the GLP-1 (9-36) composition is administered parenterally.

30. The method of claim 3, wherein the GLP-1 (9-36) composition is administered intravenously.

31. The method of claim 3, wherein the GLP-1 (9-36) composition is administered by a subcutaneous infusion pump.

32. The method of claim 3, wherein the mammal is administered at least one other treatment for inhibiting the effects of diabetes, impaired glucose tolerance, stress hyperglycemia, metabolic syndrome, and/or insulin resistance.

33. The method of claim 32, wherein the at least one other treatment is administration of insulin.

34. The method of claim 32, wherein the at least one other treatment inhibits poly(ADP-ribose) polymerase (PARP) activity or accumulation in the mammal.

35. The method of claim 34, wherein the PARP activity is inhibited by administering to the mammal a PARP inhibitor.

36. The method of claim 35, wherein the PARP inhibitor is selected from the group consisting of PJ34, 3-aminobenzamide, 4-amino-1,8-naphthalimide, 6(5H)-phenanthridinone, benzamide, INO-1001, and NU1025.

37. The method of claim 35, wherein the PARP inhibitor is selected from the group consisting of PJ34, INO-1001, and 3-aminobenzamide.

38. The method of claim 34, wherein the PARP activity is inhibited by administering to the mammal a nucleic acid or mimetic that specifically inhibits transcription or translation of the PARP gene.

39. The method of claim 38, wherein the nucleic acid or mimetic is selected from the group consisting of an antisense complementary to mRNA of the PARP gene, a ribozyme capable of specifically cleaving the mRNA of the PARP gene, and an RNAi molecule complementary to a portion of the PARP gene.

40. The method of claim 34, wherein the PARP activity is inhibited by administration of a compound that specifically binds to the PARP.

41. The method of claim 40, wherein the compound that specifically binds to the PARP is an antibody or an aptamer.

42. The method of claim 32, wherein the at least one other treatment activates transketolase in the mammal.

43. The method of claim 42, wherein transketolase is activated by administering a lipid-soluble thiamine derivative to the mammal.

44. The method of claim 43, wherein the lipid-soluble thiamine derivative is selected from the group consisting of benfotiamine, thiamine propyl disulfide, and thiamine tetrahydrofurfuryl disulfide.

45. The method of claim 32, wherein the at least one other treatment further reduces superoxide in the mammal.

46. The method of claim 45, wherein the superoxide is reduced in the mammal by administering to the mammal a compound selected from the group consisting of an α-lipoic acid, a superoxide dismutase mimetic and a catalase mimetic.

47. The method of claim 45, wherein the compound is a superoxide dismutase mimetic or a catalase mimetic selected from the group consisting of MnTBAP, ZnTBAP, SC-55858, EUK-134, M40403, AEOL 10112, AEOL 10113 and AEOL 10150.

48. The method of claim 47, wherein the compound is selected from the group consisting of M40403, MnTBAP, AEOL 10112, AEOL 10113, AEOL 10150, and ZnTBAP.

49. The method of claim 32, wherein the at least one other treatment inhibits excessive release of free fatty acids in the mammal.

50. The method of claim 49, wherein excessive release of free fatty acids is inhibited by administering to the mammal a compound selected from the group consisting of a thiazolidinedione, nicotinic acid, adiponectin and acipimox.

51. A method of inhibiting the development of disease due to diabetes, impaired glucose tolerance, stress hyperglycemia, metabolic syndrome, and/or insulin resistance in a mammal, or conditions resulting therefrom, the method comprising treating the mammal with a pharmaceutically acceptable composition comprising GLP-1 (9-36) sufficient to inhibit hyperglycemia-induced or free fatty acid-induced reactive oxygen formation in the mammal.

52. The method of claim 51, wherein the disease is an atherosclerotic, microvascular, or neurologic disease.

53. The method of claim 51, wherein the disease is selected from the group consisting of coronary disease, myocardial infarction, atherosclerotic peripheral vascular disease, cerebrovascular disease, stroke, retinopathy, renal disease, neuropathy, and cardiomyopathy.

54. The method of claim 51, wherein the mammal is administered at least one other treatment for inhibiting the effects of diabetes, impaired glucose tolerance, stress hyperglycemia, metabolic syndrome, and/or insulin resistance.

55. A method of reducing hyperglycemia-induced or free fatty acid-induced inactivation of prostacyclin synthase in a mammal, the method comprising treating the mammal with GLP-1 (9-36) sufficient to inhibit the hyperglycemia-induced or free fatty acid-induced reactive oxygen formation in the mammal.

56. The method of claim 55, wherein the mammal has or is at risk for hypoxic pulmonary hypertension.

57. The method of claim 55, wherein the mammal is at risk for undergoing an acute thrombotic event.

58. The method of claim 57, wherein the acute thrombotic event is a stroke or a heart attack.

59. A method of inhibiting hyperglycemia-induced or free fatty acid-induced decrease in endothelial nitric oxide synthetase (eNOS) activity in an endothelial cell, the method comprising treating the mammal with GLP-1 (9-36) sufficient to inhibit the hyperglycemia-induced or free fatty acid-induced decrease in eNOS activity in the cell.

60. The method of claim 59, wherein the endothelial cell is part of the vascular tissue of a living mammal.

61. The method of claim 60, wherein the living mammal has or is at risk for having diabetes, impaired glucose intolerance, stress hyperglycemia, metabolic syndrome, and/or insulin resistance.

62. The method of any one of claims 1-61, wherein the GLP-1 (9-36) is formulated in a slow release composition.

63. The method of claim 62, wherein the slow release composition is a microcrystalline composition.

64. The method of claim 62, wherein the GLP-1 (9-36) sequence is altered to form a more slow-release composition than the GLP-1 of SEQ ID NO:1.

65. The method of claim 64, wherein GLP-1 (9-36) sequence is selected from the group consisting of SEQ ID NOs:3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15 and 16.

66. The method of claim 64, wherein the GLP-1 (9-36) sequence comprises at least one acetylated lysine where the acetyl group is a myristoyl group.

67. An isolated and purified GLP-1 (9-36) consisting essentially of a sequence selected from the group consisting of SEQ ID NOs:3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, and 16.

68. The GLP-1 (9-36) of claim 67, wherein the GLP-1 (9-36) is an amide.

69. The GLP-1 (9-36) of claim 67, wherein the GLP-1 (9-36) further comprises an additional Arg at the carboxy terminus.

70. The GLP-1 (9-36) of claim 67, wherein the GLP-1 (9-36) sequence comprises at least one acetylated lysine where the acetyl group is a myristoyl group.

71. A composition comprising the GLP-1 (9-36) of claim 67 in a pharmaceutically acceptable excipient.